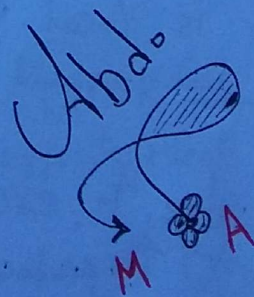


Challenge is difficult
but
The Failure in it
is more difficult



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Factors affecting Contractility & excitability & conductivity rhythmicity?

according to	excitability	rhythmicity	conductivity
1- Nervous F * sym — * para —	increase ↑ decrease ↓	increase ↑ decrease ↓	increase ↑ decrease ↓
2- physical F ↑ * Temperature	increase ↑	increase ↑ heart rate	increase ↑
3- Mechanical F	no effect	Distension of The right atrium ↑ increase	no effect
4- Chemical F * Hormone	Catecholamine ↑ Thyroxine ↑	↑	↑
* blood gas	hypoxia ↓	mild ↑ severe ↓	↓
* pH	no effect	acidosis ↓ alkalosis ↑	same
* Toxins	no effect	inhibite	same
* drugs	cholinergic drugs ↓ Xanthines ↑	digitalis ↓	digitalis ↓
* inorganic ions hyperkalima K ⁺ hyper Natrima Na ⁺ hyper Calsima Ca ²⁺	↓ no effect ↓	↓ no effect ↓	↓ ↓ ↓

* Contractility

- 1- Nervous F * symp (+ve) inotropic effect * para — (-ve) inotropic effect
- 2- physical F * temperature (+ve)
- 3- Mechanical F
- preload (Length-tension relationship) ↑
 - afterload (Force-velocity relationship) ↓
 - heart rate (Force-frequency relationship) ↑
- 4- Chemical F
- * hormone → catecholamine - Thyroxine (+ve)
 - * Blood gas → hypoxia - hypercapnia (+ve)
 - * pH → acidosis (-ve) alkalosis (+ve)
 - * Toxins → Certain snakes Venoms (-ve)
 - * drugs → digitalis and Xanthines (↑) anesthetic (-ve)

2 Myocardial action potential = Fast response Action potential

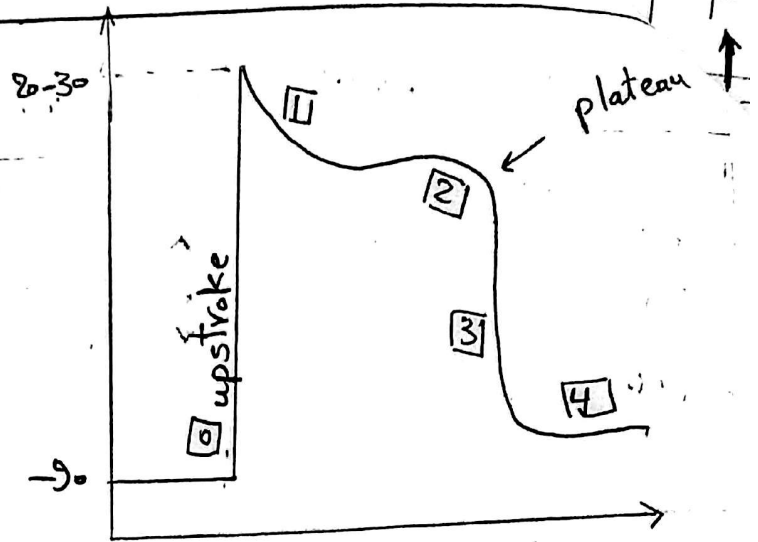
* The resting potential is about -90 mV and remain constant till excitation

* The upstroke is Rapid
It is due to Na^+ influx

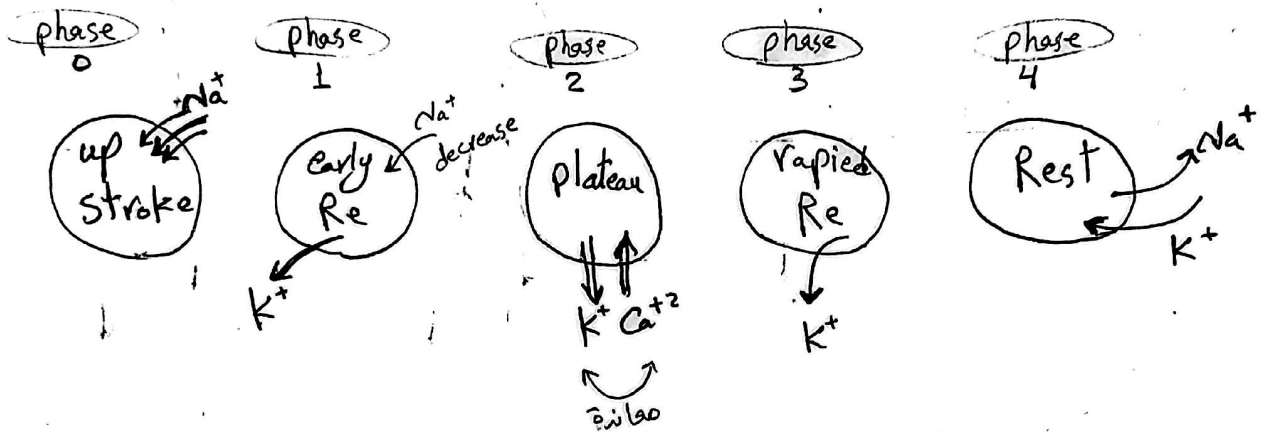
* There is prominent plateau

* There is no prepotential

* The falling phase is Rapid



→ Ionic basis of Myocardial A-P

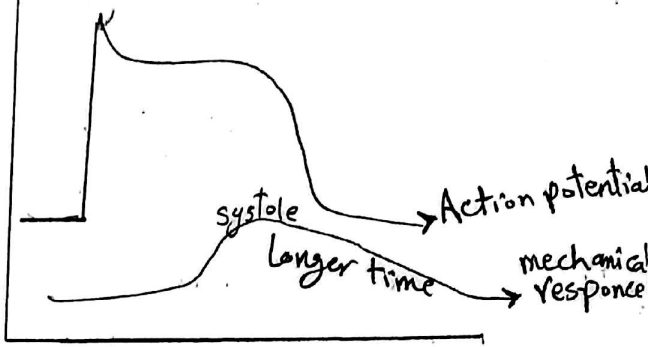


3 Excitability changes of Myocardial A-P

according to	Absolute refractory	Relative refractory	Super normal
duration	start of 0 middle 3	middle of 3 continuous 4	rest of phase 4
Effect of stimulus	No stimulus	Strong stimulus	Weak stimulus
Response	No response	Slow response	Fast response

Relation between Mechanical and excitability changes of Cardiac m

- The mechanical response starts just after the depolarization phase of action potential
- It continues for a longer time than the action potential.
- The systole reaches maximum at the end of plateau.



while The first half of diastole continues with rapid phase of repolarization.

5 Isometric contraction and relaxation phases of Cardiac cycle

	isometric contraction	isometric relaxation
① Ventricular → pressure → volume	* ↑ rises sharply to 80 mmHg * remains unchanged	* ↓ falls sharply to 5 mmHg * remain unchanged
② Valves	all valve are closed	all Valve are closed
③ sound	First heart sound	second heart sound.
④ Atrial pressure	↑ increase slightly	still increase
⑤ Aortic pressure	↓ to minimum 80 mmHg	↑ increase then gradually decrease ↓
⑥ ECG	Q wave start about 0.02s before this phase R-S wave occur during it	T wave end in this phase

6 Vagal tone & Vagal escape phenomenon

* Vagal tone phenomenon

* definition (It's continuous inhibitory effect exerted by the vagus nerves) on the heart during rest

→ while In the SA node is controlled by sympathetic tone which reducing its rhythmicity from about 100 to about 75 impulses/minute

* Mechanism of the Cardiac Vagal Tone. Follow:

- ① The initiating stimulus is (arterial blood pressure)
- ② It stimulates certain stretch receptors called arterial baroreceptors or pressoreceptor. They are located in aortic and carotid sinus.
- ③ Impulses from these receptors are conducted to Medulla by afferent nerve fiber in the aortic and carotid sinus nerve.
- ④ Such impulses inhibit the activity of the VCC.
- ⑤ It stimulates the CIC.
- ⑥ The latter, in turn, discharge tonic impulses via efferent vagal nerve fibres that depress the inherent high auto rhythmicity of the SAN.

* The Vagal Escape phenomenon

* definition (The escape of ventricles from the inhibitory effect of the Vagal tone)

- after heart beating stops by strong bilateral vagal stimulation, the ventricle starts beating after a short time by their own idioventricular rhythm (25-40/minute).
- This property is beneficial in cases of Complete Heart block.

7. Regulation of Cardiac output & Heart rate & Arterial blood pressure & Arterioles diameter?

* Regulation of CO follows 2 Mechanism

1. Extrinsic regulation

* definition (This modifies both the stroke volume & heart rate by effect of autonomic nerves and certain hormone)

1- role of autonomic nerve

- * Sympathetic ↑ Cardiac output
- * Para — ↓ Cardiac output

2- role of Certain Hormone

- * Catecholamines
 - * Thyroxine
 - * Glucagon
- ↑ Cardiac output

Intrinsic Regulation

* definition (The modifies The stroke volume only and It include heterometric and homometric)

	Heterometric	homometric
1- stimulus	increased <u>EDV</u>	increased aortic impedance
2- basic	Starling law	change in inotropic state
3- Duration	short term	long term
4- timing	before homometric	After heterometric
5- loading state	preload phenomenon	Afterload phenomenon
6- limit	Certain level of <u>EDV</u>	available Ca^{+2} in cardiac muscle
7- Significance	balancing CO of both ventricular	steady $\uparrow$ in cardiac contractility $\uparrow$ ABP

* regulation of HR

1 Nervous regulation of The heart rate

The HR is nervously regulated by cardiovascular centers which is influenced by The supraspinal centres & many reflexes

(A) Supraspinal center

- 1- Cerebral Cortex
- 3- The Respiratory centre

2- hypothalamus and limbic system

(B) Reflexes From CVS

- 1- Marcy's Reflex
- 2- Bainbridge Reflex

- 3- Coronary stretch reflex
- 4- Bezold-Jarisch reflex

(C) Reflexes From Extravascular system

- 1- From Lunge $\rightarrow$ pulmonary stretch reflex
 $\rightarrow$ pulmonary chemo reflex
- 2- From skeletal muscle
- 3- from skin and viscera
- 4- from Eyes

2 Chemical regulation of The heart rate

(A) Effect of changes in blood gases

* hypoxia $\rightarrow$ mild $\uparrow$ HR
 $\rightarrow$ severe $\downarrow$ HR

* Hypercapnia $\uparrow$ HR

- Hypoxia ↑ by ③ mechanism
- stimulating SA node
 - inhibiting CIC
 - stimulating VCC
- Hypercapnia ↑ HR by ② mechanism
- 1- inhibiting CIC
 - 2- stimulating VCC through peripheral and central Chemoreceptor

② Effect of Hormones and Drugs

- 1- Adrenaline
- small doses ↑ HR by action on β_1 receptor in SA
 - large doses ↓ HR by Meary's reflex

2- (Nor) adrenaline ↓ HR by Meary's Reflex

3- Thyroxin ↑ HR by stimulating SAN

4- Atropine ↑ HR by blocking parasymp

5- Histamine ↑ HR by Meary's Reflex

6- Bile salt ↓ HR by stimulate CIC

* Regulation of ABP Follow by 3 mechanism

① Short term mechanism

- 1- Arterial baroreflex start by stimulation of baroreceptor when ↑ ABP
- 2- Arterial Chemoreflex " " " chemoreceptor when ↓ ABP
- 3- The CNS ischaemic response occurs when ↓ ABP
- 4- The abdominal compression reflex by increase skeletal muscle tone ↑ ABP

② Intermediate term mechanism

- 1- Capillary Fluid shift ↓ ABP by increase blood volume
- 2- Stress relaxation ↑ ABP by increase tension in wall of Arteries
- 3- Renin - angiotensin V.C ↓ ABP by secrete renin hormone
- 4- Right atrial mechanism ↓ ABP by increase blood volume

③ Long term mechanism

This mechanism control ABP by excretion of water and salt by The Kidney This occur by

- a) glomerular Filtration
- b) secretion of aldosterone hormone

Regulation of Arteriolar diameter

The arteriolar diameter is regulated by both Local and Systemic Factors

A Local Factor

- 1- O_2 supply O_2 lack lead to arteriolar VD
- 2 Metabolites lead to arteriolar VD
- 3- Local temperature $\uparrow$ temperature lead to arteriolar VD
- 4- Autoregulation lead to arteriolar VD
- 5- Locally-released arteriolar regulators
 - * Serotonin - Thromboxane - Endothelins $\rightarrow$ VC
 - * prostaglandin $I_2 \rightarrow$ VD

B Central or systemic factor These factor include

1- Hormonal regulation

A Circulating VC substance

- catecholamines
- Vaso pressin
- angiotensin
- Serotonin

B Circulating VD

- ANP
- VIP
- Histamine
- Kinins

2- Neural Regulation

A VC nerves $\rightarrow$ Sympathetic noradrenalin nerves except at coronary vessels

B VD nerves $\rightarrow$ 1- somatic
2- Para
3- coronary vessels in symp

8 Factors maintaining ABP

① Cardiac output The ABP is generally directly proportionate to CO
* Changes in The SV affect mainly systolic pressure while changes in The HF affect mainly diastolic

② Peripheral Resistance PR is essential For maintaining ABP particularly diastolic pressure

③ Elasticity of aorta and large A

* This is essential For production and mainteny of a relatively high diastolic BP It's effect is called windkessel effect

④ Blood volume and Circulatory capacity

* These parameters determine ABP by controlling The Mean systemic filling pressure. MSFP varies directly with ABP

9] Factor affecting peripheral resistance & Venous return

* Factor affecting of PR

- 1- The diameter of The blood vessel $\propto \frac{1}{PR}$
 - 2- The length of The blood vessel $\propto PR$
 - 3- The blood viscosity $\propto PR$
- ⇒ **PR** is directly proportionate to The length of BV and The blood viscosity and inversely proportionate to The diameter of BV
- ⇒ The diameter of BV remain The most important factor For PR

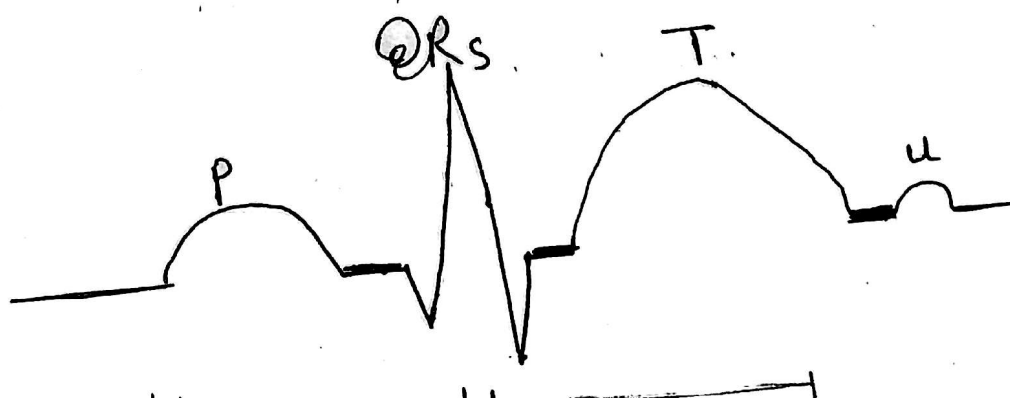
* Factor affecting of VR

- 1- Venous pressure gradient
- 2- skeletal muscle **pump**
- 3- Respiratory **pump**
- 4- Venous tone
- 5- Cardiac suction Force
- 6- Blood Volume
- 7- gravity
- 8- Arteriolar and capillary diameters
- 9- Sympathetic stimulation

10] ECG Waves & intervals & segments

* **Waves** P Q R S T and There are **U waves** but little

	P	QRS	T	U
represent	atrial <u>depolar</u>	Ventricular <u>depolar</u>	Ventricular <u>repolar</u>	Slow repolarization of Capillary muscle
Duration	0.1 s	0.1 s	0.25 s	
Voltage	0.25 mv	1 mv	0.5 mv	It's prominent in hypokalemia



Intervals

	P-R interval	Q-T interval
measure	From begin P → beginning QRS	beginning QRS to end T
Represent	Conduction from SAN → ventricle	ventricular dep & repolarization
Duration	0.12 — 0.21 s	0.34 — 0.43 s
abnormality	Prolonged in ① A-V block ② Vagal stimulation Shortened in ① A-V nodal rhythm ② Symp — stimulation	shortened in ↑ heart rate

* segments

S-T segment	T-P segment	P-R segment
→ measure From The end of (QRS) to The beginning of (T)	→ measure From end of (T) till The start (P)	
⇒ represent all ventricular muscle are depolarized ⇒ elevated in ischemia — infarction	⇒ It's The true isoelectric line	

11 Anging pectoris & Myocardial infarction & atrial Flutter atrial fibrillation & heart block

* Anging pectoris i.e ischemia and injury

(Manifestation of ischemia)

The resulting hypoxia retards repolarization in The epicardial side of The affected part so repolarization starts from endocardial toward The epicardium producing inverted T waves

(Manifestation of injury)

This causes S-T segment shift and current of injury i.e Flow of current The pathologically depolarization and The normal parts The current of injury lead to incomplete repolarization of Cardia muscle



* Myocardial infarction i.e. necrosis

* This occurs almost always in the left ventricular wall or interventricular septum or both

* In This Case

→ Myocardium becomes electrically-inert and can't be depolarized

→ appear hole or window in left ventricular wall

→ appear Pathological Q wave more than 0.2mv and become permanent for life

∞ Ischemia and injury and necrosis occur due to coronary heart diseases

* atrial Flutter

* definition (This is a pathological condition in which the atria beat regularly at a rate of 250-350/minute by impulses discharged from single ectopic focus)

⇒ The A-V node can't conduct more than 230 impulses/minute

⇒ occur in complete heart block

⇒ The ventricles respond once for every 2 or 3 atrial beats

⇒ ECG

* each 2 or 3 P wave are followed by one QRS

* P wave are abnormal

* QRS and T waves are normal

* atrial Fibrillation

* definition (This is a pathological condition in which the atria beat irregularly at rate of 350-500/minute by impulses discharged from multiple ectopic focus)

⇒ occur physiological heart block but irregularly

⇒ It prevents development of ventricular fibrillation so ventricles beat irregularly at rate of 100-150/minute and occur pulse deficit

⇒ ECG

* There are (no) P wave

* QRS and T wave are normal

* appear F wave called fine wave
It has no effect on life

Heart block

* definition (failure of conduction of impulses from The S-A node to The ventricle)

- * Type
- ① S-A block
 - ② A-V block
 - ③ bundle-branch block

1 Sino atrial block

* In This Case impulses are produced in The active Focus in S-A node but are not conducted Through The nodal tissue to atrial muscle

- when S-A block occurs The A-V block takeover The heart pacemaker
- ECG shows cycles without P waves

2 Atrio ventricular block

According A-V block is clinically classified (3 type or 3 degree)

① First degree

* In This Case ⇒ The conduction time A-V node and bundle is prolonged more Than normal

⇒ All atrial impulses still reach The ventricles but with a longer delay

② Second degree

This is partial block in which some atrial impulses are conducted to ventricles and some are not

There are 2 type

- ① regular → If There is Fixed ratio between atrial impulses and reaching to ventricles
- ② irregular → If There is No Fixed ratio " " " "

③ Third degree

This is complete block in which No impulse arising in atria can reach to ventricle

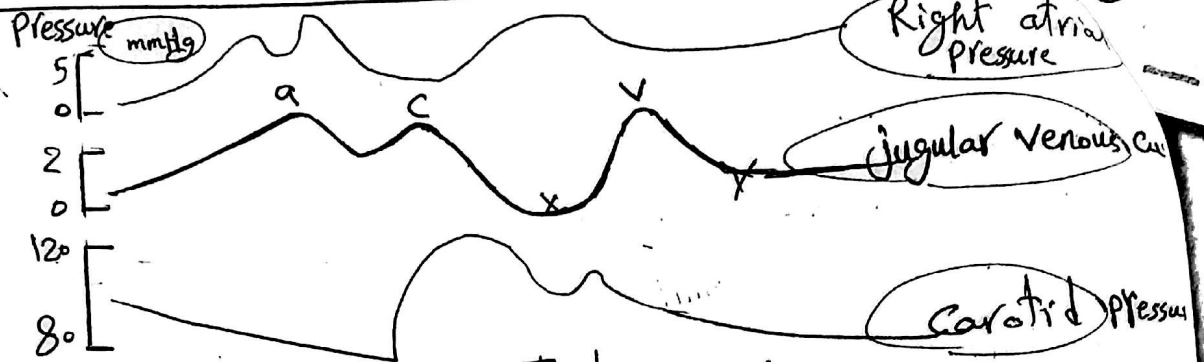
3 Bundle-branch block (BBB)

* In This Case

→ Impulses Cannot be conducted Through Left branch (LBBB) or Right branch (RBBB)

→ The ventricle with normal branch will beat earlier Than blocked branch

12] jugular pulse Curve



* definition (This is the pressure changes that occur in the jugular veins during cardiac cycle)

* normal composition 3 positive waves called a c v waves
2 depressions called x y descents

"a" wave This wave occurs during atrial systole

→ Its ascending limb is due to

- ① increase of the right atrial pressure
- ② accumulation of blood in jugular veins

"c" wave This wave occurs during isometric ventricular contraction

→ Its ascending limb is due to

- ① bulging of the tricuspid valve into the right atrium
- ② carotid artery pulsations

* on other hand x descents occur during early ejection due to

- ① atrial relaxation
- ② pulling of A-V ring downward during ventricular systole
- ③ Return of the tricuspid valve to normal position

"v" wave This wave occurs during ① most of ejection phases
② isometric relaxation phase

→ Its ascending limb is due to ↑ atrial pressure as result of

- ① accumulation of blood of venous return in atria
- ② upward A-V ring after vent-systole

* on other hand y descents occur during rapid filling phase due to opening A-V valve

Clinical Significance of Jugular pulse curve

(A) a-c interval (From a wave to c wave) It's normal duration is 0.12 - 0.21 second - It corresponds to (P-R) interval of ECG

(B) The waves of curve are altered in many cardiac disease

eg ① large (C-V) are recorded in case of tricuspid incompetence

② (a) wave amplitude is much increased in cases of 1-tricuspid stenosis
2-pulmonary stenosis
3- " hypertension

③ giant (a) wave called Cannon waves are recorded if the right atrium and right ventricle contact at the same time

④ (a) waves disappear in atrial fibrillation

⑤ by jugular venous pressure (JVP) can differentiate atrial and ventricular extrasystoles because disappear in vent - extrasystoles

13 Heart Sound

* Compare between 1st 2nd

	according to	First heart sound	second heart sound
①	Causes	Closure of The A-V valves	Closure of The semilunar valves
②	pitch	Low pitched sound $f = 25-40 \text{ Hz}$	It higher pitch Than first sound.
③	Quality	Soft	Sharp
④	Timing	At The onset of Ventricular (systole)	At The onset of Ventricular (diastole)
⑤	Duration	0.15 second	0.1 second
⑥	Loudness	Fainter Than second sound (base)	Louder Than first sound (apex)
⑦	Site of hearing	⇒ Mitral in The Fifth left intercostal space at mid clavicular line ⇒ Tricuspid in The Fourth left intercostal space near The sternum	⇒ Aortic The second (right) intercostal space near sternum ⇒ Pulmonary The second (Left) " " " " "

* Third heart sound

- Timing During The rapid Filling phase
- Cause Vibrations set up by rush of blood from atria to ventricle During early diastole
- Duration 0.1 second
- Characters It is a soft and very low pitched
- hearing During expiration at The apex for Filling The left ventricle

* Fourth heart sound

- Timing During atrial systole
- Cause Vibration set up by rush of blood from atria to ventricle During atrial systole
- Duration 0.03 — 0.05 s
- Characters It is faint and very low pitched
- hearing at apex of The heart

14 Edema " definition — Causes — types "

* definition (It's accumulation of excess Fluid in The body)

* Type ① Intracellular edema
② Extracellular edema

1. Intracellular edema This is produced as result of

- ① Depression of cell membrane metabolic activity due to 1- ischemia
2- O₂ lack
- ② Inflammation This increase The cell membrane permeability which allow Na⁺ and other ions to diffuse into The cells

2. Extracellular edema This is accumulation of excessive amounts of interstitial Fluid It has 2 main causes

- ① Excessive leakage of Fluid From The capillaries due to
 - a- increase of The capillary hydrostatic pressure
 - b- hypoproteinemia i.e ↓ synthesis of plasma protein
 - c- increase of capillary permeability i.e ↑ filtration of both The tissue Fluid and plasma protein
 - d- Salt & water retention specially in renal disease i.e ↑ blood volume and capillary pressure

inadequate lymph drainage

* This is due to blockage of The lymphatic vessels

e.g 1- Cancer cells

2- Filaria worms → which causes The elephantiasis disease

* The edema in this case is non-pitting and called lymphedema

15 Starling Law & Mary's Law & Bainbridge reflex

* Starling law

This Law describes (Length-tension relationship)

It state That (The Force of Myocardial contraction is directly proportional to The initial length of Cardiac muscle)

* Significance of The starling law

1 In normal hearts

The starling law allow matching of The ventricular output to changes in venous return and maintains The balance between The outputs of both ventricles

2 If The aortic pressure ↑ lead to

- ① decrease stroke volume of left vent
- ② Forceful contraction
- ③ accumulated blood ejects in left ventricle

3 In trasplanted hearts

In These hearts (heterometric autoregulation of Cardiac Contractility is The principal mechanism) That adjusts The pumping capacity of The heart

4 In failing heart

The EDV ↑ due to weak ventricular contraction and according to starling law This tends to ↑ increase The Force of contraction

* Mary's Law

It state That

(A rise of The arterial blood pressure ABP ↑ lead to decrease of The heart rate HR ↓ and Vice versa provided other factors affecting The heart rate remain constant)

* It is initiated by stimulation of the (arterial baroreceptors)
This affects
① stimulation of both the CIC and VDC
② inhibition of the respiratory centre
③ inhibition of secretion of the anti diuretic hormone

* Carotid sinus syndrome is abnormally hypersensitive
lead to
① brady cardia
② ↓ cardiac output
③ brain ischemia and syncope

* Brainbridge Reflex

i.e (an increase in right atrial pressure lead to heart ^{acceleration} acceleration)

* impulses are discharged mostly from type A atrial receptor
via afferent → to Medulla → stimulate the VCC
(However) such response is (not) constant

* Effect of ↑ right atrial pressure

- 1- acceleration of the heart rate = Bainbridge Reflex
- 2- " of breathing = Harrison reflex
- 3- Generalized VD and reduction of ABP
- 4- Coronary VD
- 5- inhibition of secretion of anti-diuretic hormone (ADH)

16 Shock and Hemorrhage

* Shock

* definition (Clinical syndrome of circulatory failure characterized by a low cardiac output, hypotension)

- * Type
- ① hypovolemic shock (cold shock)
 - ② Distributive shock (warm shock)
 - ③ Cardiogenic shock
 - ④ obstructive shock

hypo Volamic shock

* This occurs as result of (severe reduction of blood volume)

* Its manifestations

- 1- rapid Thready pulse
- 2- Tachypnea
- 3- Thirst
- 4- Cold pale skin

* Its main Causes

- 1- Severe haemorrhage
- 2- Severe Trauma
- 3- Extensive burns
- 4- Major surgery
- 5- Dehydration

2 Distributive shock

* This occurs as a result of (widespread V.D of blood vessels)

* Its manifestations same hypovolamic but warm skin

* Its main Causes

- 1- Strong Emotions
- 2- sever allergic reactions
- 3- Bacterial reaction

3 Cardiogenic shock

* This occurs as a result of (inadequate pumping action of Heart)

* Its manifestations same other Types + Congestion of The lungs and Viscera

* Its main Causes

- 1- extensive Myocardial infarction
- 2- Acute Myocarditis
- 3- Heart Failure
- 4- severe ventricular arrhythmias

4 obstructive shock

* This occurs as a result of (obstructive of blood Flow in The lung and heart)

* Its main Causes

- 1- large pneumothorax
- 2- Massive pulmonary embolism
- 3- Cardiac tumor
- 4- Cardiac tamponade

Dangers of Shock. How does it lead to death?

→ Death occurs a result of positive Feedback cycles

Hypotension → ischemia → low Cardiac output → more hypotension → death

⇒ Irreversible shock (refractory shock)

* definition (terminal Fatal stage of shock It occurs particularly in hypovolemic shock, depending largely on the lost blood volume)

* How does it occur?

⇒ As a result of spasm of pre capillary sphincters and the venules
⇒ when ↓ capillary perfusion lead to 1- hypoxic tissue damage
2- relaxation of these sphincter

(Treatment of shock)

① general measures in all shocks

① giving vasopressor agents

② Keeping the patient in the recumbent position and raising the foot of bed

② specific measures

a - Blood transfusion in haemorrhagic and traumatic and surgical

b - Adrenaline in anaphylactic shock

c - Dopamine in cardiogenic shock

d - Antibiotic in septic shock

* haemorrhage

* definition (It is loss of blood from CVs)

* Manifestations of Haemorrhage

1- rapid weak pulse

2- rapid respiration

3- Thirst

4- pale - cold - clammy skin

5- Excessive sweating

6- oliguria or anuria

7- restlessness

8- acidosis

* Compensatory Reactions to acute Haemorrhage

① rapid Compensatory Reactions

→ reactions that correct the hypovolemia

① capillary fluid shift

② contraction of the splenic capsule

③ Mobilization of the labile tissue protein into the blood.

Reaction that increase Cardiac output & Peripheral resistance

- 1- inhibitory effect of arterial baroreflex
- 2- stimulation arterial chemoreflex
- 3- secretion of catecholamine

A Delayed compensatory reactions

→ reaction That maintain a high peripheral resistance

- 1- stimulation of secretion of antidiuretic hormone (ADH)
- 2- formation of angiotensin II
- 3- increased tension in arteriolar walls

→ reaction That maintain a normal blood volume

- 1- restoration of plasma volume
- 2- restoration of plasma protein

17 Factor affecting (Coronary blood Flow — Local blood Flow Capillary pressure — pulmonary blood Flow)

* Factor affecting Coronary blood Flow CBF

① (Mechanical Factors)

during diastole → Intramyocardial pressure → ↓ CBF

1- The CBF undergoes changes during The cardiac cycle

* occur most of CBF during diastole

* being maximal at The end of The isometric relaxation phase

2- The CBF is affected by changes in The heart rate

In cases of Tachycardia → ↓ The CBF

② (Metabolic Factors)

due to The CBF is directly related to The activity of The heart

↑ CBF → high activity

↓ CBF → low activity

③ (Nervous factors)

* stimulation of The cardiac symp. → ↑ CBF → C.V

* Parasymp. stimulation → ↓ CBF → C.D

④ (Hormonal factors)

* catecholamines → ↑ CBF → stimulation beta receptor → ↑ Cardiac metabolic activity

* Vaso pressin → ↓ CBF Through V.C of Coronary

* Regulation (factor affecting) of Local blood flow It's follow

① Short-term Regulatory mechanisms

- ⇒ These are the mechanisms that regulate the arteriolar diameter
- ⇒ They involve rapid changes in arteriolar diameter within ^{sec} to maintain optimum local blood flow rate

② Long-term Regulatory Mechanisms

- ⇒ These involve slow changes in the degree of vascularity of tissue within ^{days} → ^{month}
- ⇒ These changes include:

① Angiogenesis = growth of new blood vessels

- * This occurs in response to certain factors that are released from ischaemia tissues or tumours

② Development of collateral circulation

- * This occurs when an artery or a vein is blocked within 1-2 minutes after blockage

* Factor affecting of Capillary pressure (CP)

① Pre-Capillary Factors

- ① pre-capillary resistance if it increase $\uparrow$ $\xrightarrow{\text{inverse}}$ $\downarrow$ CP
- ② Arterial blood pressure if it increase $\uparrow$ $\xrightarrow{\text{directly}}$ $\uparrow$ CP

② Post-Capillary Factors

- ① post-capillary resistance if it increase $\uparrow$ $\xrightarrow{\text{directly}}$ $\uparrow$ CP
- ② Venous pressure if it increase $\uparrow$ $\xrightarrow{\text{directly}}$ $\uparrow$ CP

* Factor affecting of pulmonary blood flow

① pulmonary vascular resistance (PVR)

- ⇒ factors that increase PVR → increase the pulmonary arterial blood pressure and vice versa

① Nervous factors

- * Symp. stimulation → $\uparrow$ PVR and $\uparrow$ pulmonary arterial B.P
- * Para. stimulation → opposite effect

② Chemical factors

- * angiotensin II - endothelin - thromboxan → $\uparrow$ PVR and $\uparrow$ "
- * histamine - bradykinin → opposite effect

Systemic hypoxia → ↑ PVR and ↑ pulmonary arterial BP

4 Respiratory movement

- * During inspiration → ↓ PVR and ↓ " "
- * During Expiration → opposite effect

5 Lung diseases eg emphysema - pulmonary fibrosis and embolism ↓ PVR and ↓

6 Left atria pressure

Rise of The Left atria pressure → ↑ pulmonary arterial BP

7 Cardiac output

Increase in The Cardiac output → small rise of " "

18 Triple response

* definition (This is response that commonly occurs in The skin as a result of either injury or inflammation or insect bites)

* It consists of 3 reactions that occur successively

① Red reaction

- * This appears in about 10 seconds at The site of The stroke
- * It is due to Capillary dilatation

② Spreading flare

- * This is diffuse mottled reddening that first appears within about half a minute around The area of injury Then gradually spreads outwards

* It is due to arteriolar VD which is produced by The antidromic Local axon reflex mechanism

③ Swelling

- * This is local edema that appears within few minutes around The area of injury

* It is due to excessive extravasation of fluid produced by 1- Histamine
2- Substance P